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Thoracic Spinal Cord Compression Precipitated by Epidural Steroid Injection Due to Undiagnosed Dural Arteriovenous Fistula – A Case Report

Stephen N. Dunay, MD[1], John Wainwright, MD[2], Ammar Siddiqui, MD[3], and Gurmeen Kaur, MBBS[4]

Background: Spinal dural arteriovenous fistula is an abnormal connection between the arteries and veins surrounding the spinal cord. Symptoms may be vague contributing to its diagnostic difficulty. In some cases, patients may develop lower extremity weakness or sensory deficits, back or leg pain, and bowel or bladder dysfunction.

Case Report: We present a case of a female patient with chronic back pain who underwent routine and elective interlaminar epidural steroid injection and developed symptoms of thoracic cord compression due to a thoracic dural arteriovenous fistula that had not been previously identified on imaging.

Conclusions: Our case emphasizes the importance of early recognition of symptoms of acute cord compression, as well as close review of magnetic resonance imaging in order to identify subtle changes that may indicate a rare anatomical anomaly that can cause significant disability.

Key words: Epidural, ateriovenous fistula, cauda equina, spinal cord compression

BACKGROUND

Spinal dural arteriovenous fistula (SDAVF), a rare anomaly, is an abnormal connection between the arteries and veins surrounding the spinal cord (1,4). Their pathophysiology is unclear, but they are thought to be acquired (2), and they are more common in men > 50 years old. These are the most commonly discovered vascular malformations of the spinal cord and a treatable cause of progression to para- or tetraplegia (3).

The arteries that supply the spinal cord eventually form meningeal arteries (2). The location at which a meningeal artery pierces the dura represents a possible site for the development of an SDAVF, and they most commonly affect the thoracic and lumbar spine (2,4). Although frequently asymptomatic, symptoms may be vague, which contributes to its diagnostic difficulty. In some cases, patients may develop lower extremity weakness or sensory deficits, back or leg pain, and bowel or bladder dysfunction. The diagnosis of this anomaly is further complicated by the fact that small SDAVFs are not always clearly seen on magnetic resonance imaging (MRI) without angiography (1).

CASE PRESENTATION

The patient was a 76-year-old woman with a history of third-degree heart block and an MRI conditional cardiac pacemaker, interstitial lung disease on methotrexate and prednisone, and type 2 diabetes mellitus. She was seen in the chronic pain clinic at Westchester Medical Center in Valhalla, NY for chronic low back pain, which had been getting worse with ambulation and was attributed to neurogenic claudication. At

From: [1]Interdisciplinary Pain Management, Westchester Medical Center, Valhalla, NY; [2]Department of Neurosurgery, Westchester Medical Center, Valhalla, NY; [3]Department of Anesthesiology, Westchester Medical Center, Valhalla, NY; [4]Department of Neurology, Westchester Medical Center, Valhalla, NY

Corresponding Author: Stephen N. Dunay, MD, E-mail: stephen.dunay@gmail.com
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baseline, she ambulated with pain and used a walker. MRI lumbosacral showed severe canal stenosis at L3/L4 and L4/L5 but only mild stenosis at L5/S1. On August 8, 2024, an interlaminar epidural steroid injection (ESI) was performed without any immediate complications using a 20G Tuohy needle at the L5/S1 spinal level. A mixture comprising 40 mg of Kenalog with 3 mL of preservative-free normal saline was injected (Fig. 1). There was no pain or paresthesia on injection. The patient reported significant improvement in her pain immediately after the procedure and was discharged home after an observation period of 30 minutes. She did not have any new neurological deficits immediately following the procedure and was able to ambulate out of the hospital with the assistance of her walker.

During the evening following ESI and around 7 pm, she stated to her partner that she was unable to get herself up from her reclining chair. A wheelchair was used to get her into bed, and she went to sleep. In the early morning, she awoke to use the bathroom, but she continued to not be able to move her legs nor could she urinate. Emergency medical services transported her to a nearby hospital for evaluation, and the patient's partner notified her pain specialist of her symptoms. At the outside facility, a Foley catheter was placed and 1,500 mL of urine was drained. A stat MRI was recommended but could not be performed at the outside facility because of the patient's pacemaker, and she was transferred to our facility. The Boston Scientific (Marlborough, MA) representative was notified of her transfer in order to verify the model of her pacemaker and its MRI compatibility.

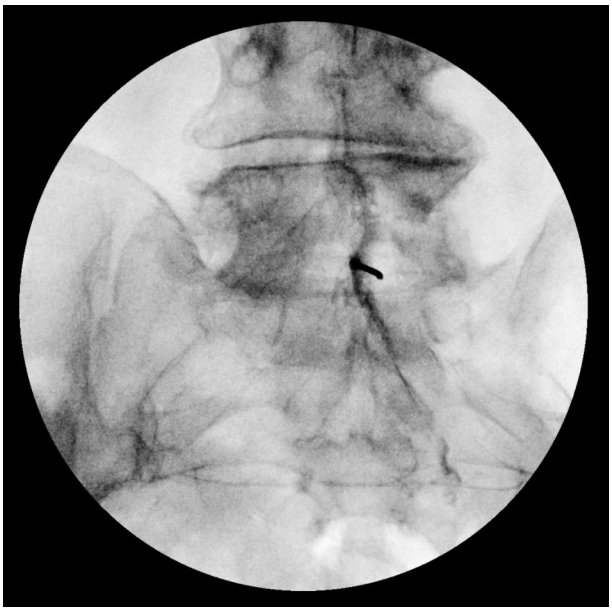

Fig. 1. Epidurogram following L5/S1 paramedian interlaminar epidural and showing normal contrast spread.

Upon arrival to the emergency department of our facility, she was promptly evaluated by the neurosurgical team. During their assessment, the patient noted ongoing numbness in both legs, without back or leg pain, and states that she felt continuing weakness but could move her legs more than she could the previous evening. Her neurological exam at the time of initial assessment revealed a decrease in rectal tone with squeeze, without saddle anesthesia, decreased sensation to light touch in both lower extremities, but more notably along the proximal aspect of the legs distally. Motor strength in the lower extremities was as follows:

- Right: 4/5 hip extensors, 2/5 hip flexors, 4/5 knee extensors, 5/5 knee flexors, 4/5 dorsiflexion and plantarflexion of the foot
- Left: 4/5 hip extensors, 2/5 hip flexors, 4/5 knee extensors, 3/5 knee flexors, 4/5 dorsiflexion and plantarflexion of the foot

The patient's exam, and recent history, was highly concerning for cauda equina syndrome possibly due to epidural hematoma in the setting of recent ESI, and a stat MRI of the lumbar spine was recommended.

An MRI without contrast of the lumbar spine showed "[n]umerous serpiginous flow voids along the dorsal aspect of the spinal canal abutting the dorsal aspect of the cord, concerning for dural arteriovenous fistula, with associated T2/STIR hyperintensity with associated diffusion restriction along the distal spinal cord at the levels of the T9-L1 which may represent chronic venous congestion/ischemia." Additionally, there were "[e]xtensive multilevel degenerative changes of the lumbosacral spine with severe canal stenosis at L4-L5 and moderate to severe canal stenosis at L3-L4" (Fig. 2). A spinal angiogram showed "[p]rimary supply to a spinal dural arteriovenous fistula by a branch arising from the right T12 segmental spinal artery. The fistulization point is identified and the early venous drainage is visualized into a tortuous congested network of the spinal cord veins" (Figs. 3 and 4).

Because of the rapid onset of her symptoms, the neurosurgical team recommended emergent decompression and she was brought to the operating room for complete laminectomy of L4 and L5, partial laminectomy of L3 and S1, and bilateral L2/L3 hemilaminotomies.

On postoperative day one following surgery, she had some improvement in motor strength and her urinary continence improved. She said that her right leg continued to be weaker than the left. She was admitted to inpatient acute rehab for treatment of ongoing deficits in mobility and activities of daily living but continued to have difficulty with balance and ambulation.

DISCUSSION

Lower extremity weakness following lumbar ESIs is a feared symptom. Possible causes for this may include neuropraxia from injectate-mediated nerve root compression, cauda equina syndrome from either the injectate itself, or a hematoma/abscess. This case was atypical as the patient did not develop immediate neurological symptoms (as would be expected with neuropraxia), nor back pain (as would be expected with cauda equina or a hematoma). The onset of her weakness was gradual, over the course of 8 hours and affected proximal myotomes rather than distal ones (as would be expected with an L5/S1 hematoma).

Her symptoms were believed to be due to a combination of cauda equina syndrome and venous congestion due to the SDAVF of the spine. This SDAVF went unrecognized initially possibly due to an absence of axial cuts on the MRI, and only after the patient developed postprocedural symptoms was the anomaly of the thoracic spine uncovered. This highlights the need for careful attention to nonspecific findings on imaging (1).

An SDAVF appears as a hyperintense lesion on T2-weighted images, and a corresponding hypointense signal can sometimes be found on T1-weighted images (2). On MRI, a combination of cord edema, perimedullary dilated vessels, and cord enhancement is characteristic (3). Similar MRI findings may be seen in cases of demyelinating conditions, infarction, inflammation, or tumor. Small SDAVFs are not always clearly seen on MRI, and spinal angiography is the gold standard imaging modality for diagnosis (1).

Treatment modalities for SDAVF include arterial embolization and surgical resection. Surgical resection results in a lower rate of recurrence but carries more risk than embolization (1,2). Surgical treatment involves coagulating or excising the dural fistula. This results in intradural interruption of the draining vein, thereby decreasing venous congestion (5). In patient discussed in this case report, open surgical resection was considered. She ultimately went on to have an uncomplicated embolization using a combination of coil and glue of the right T12 DAVF. After coiling, she regained normal motor function of the left lower extremity, and right lower extremity strength remained limited with gravity removed.

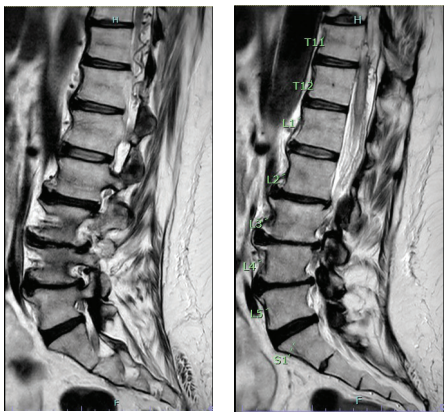

Fig. 2. Sagittal T2 weighted MRI image of the lumbar spine showing spinal canal stenosis of L3/4 and L4/5

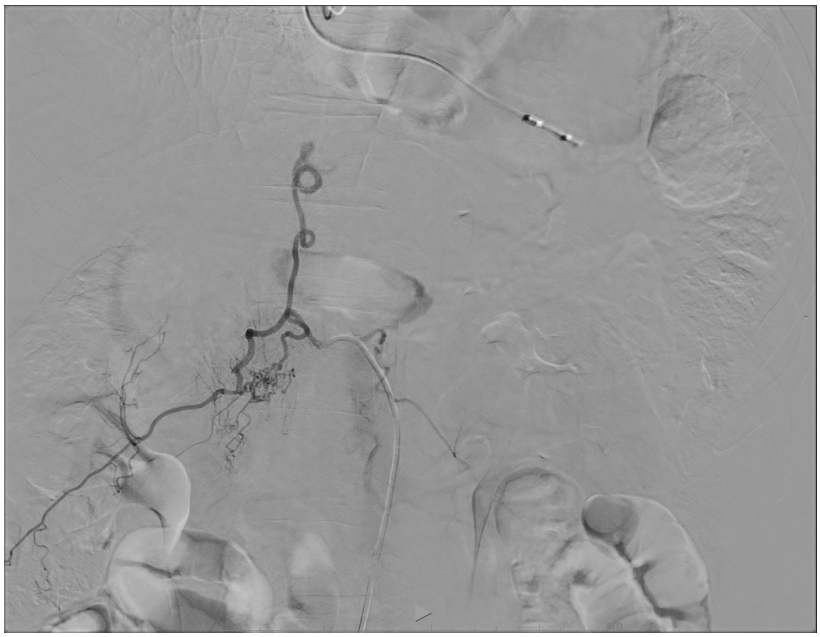

Fig. 3. Spinal angiogram showing spinal dural AV fistula arising from the T12 segmental spinal artery

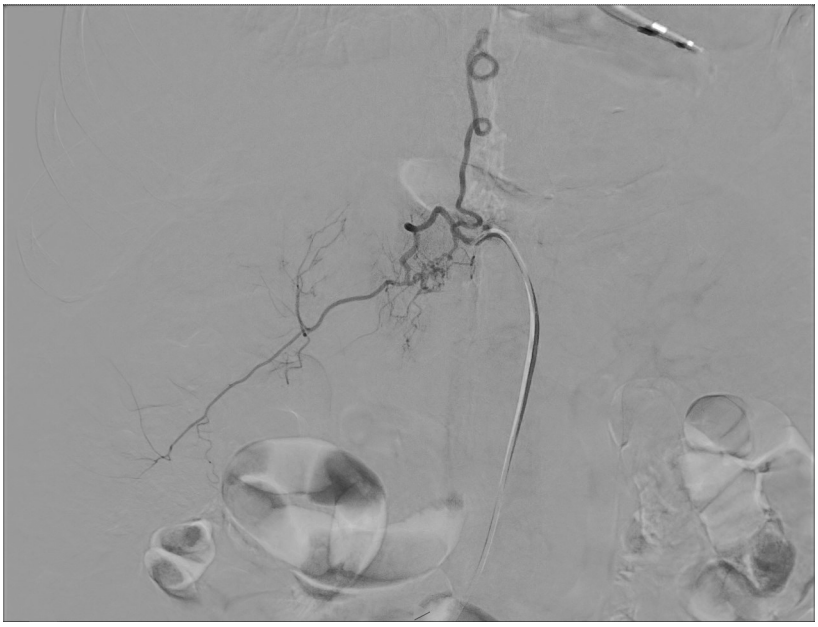

Fig. 4. Spinal angiogram showing spinal dural AV fistula arising from the T12 segmental spinal artery

CONCLUSIONS

This case highlights the importance of early recognition of a rare but potentially devastating condition exacerbated by a commonly performed pain procedure, ESI. The injection of a high volume of fluid into the epidural space of the spinal canal may cause severe neurological complications, especially when an anatomical or pathological condition in the spinal canal exists (6). If a spinal anomaly is present, injection of just 10 mL of fluids may increase epidural pressure to > 100 mmHg (6). Cauda equina syndrome and neuropraxia are more common diagnoses in patients who develop weakness following ESI; however, SDAVF should also be considered in the differential diagnosis of patients with symptoms suggestive of progressive myelopathy (2). A broad differential and thorough neurological exam is essential in the evaluation of patients with new neurological complaints following a pain procedure, such as ESI. Full recovery is more likely in patients in whom there is early recognition of the symptoms of cord compression, comprehensive examination and appropriate imaging, and prompt referral to a neurosurgeon for definitive treatment (1).

Human Ethics

Written informed consent was obtained from the patient for the publication of this case report, including details of their medical history and treatment. The patient understood that while efforts would be made to anonymize their information, complete confidentiality could not be guaranteed.

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